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NEWS	6	FEB 16	New FASTA Display Formats Added to USGENE and PCTGEN
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NEWS	8	FEB 16	INSPEC Adding Its Own IPC codes and Author's E-mail Addresses
NEWS	9	APR 02	CAS Registry Number Crossover Limits Increased to 500,000 in Key STN Databases
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NEWS	11	APR 02	DWPI: New display format ALLSTR available
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=> S mo25 or (Mouse protein 25)
L1          393 MO25 OR (MOUSE PROTEIN 25)

=> s l1 and Miyamoto
L2            0 L1 AND MIYAMOTO

=> s l1 and Miyamoto.au.
L3            0 L1 AND MIYAMOTO.AU.

=> s mo25 and (Mouse protein 25)
L4           38 MO25 AND (MOUSE PROTEIN 25)
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=> duplicate
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ENTER L# LIST OR (END):l4
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ESBIOBASE, WPIDS, BIOENG'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L4
L5            15 DUPLICATE REMOVE L4 (23 DUPLICATES REMOVED)

=> d l5 1-15 bib

L5   ANSWER 1 OF 15  EMBASE  COPYRIGHT (c) 2010 Elsevier B.V. All rights
     reserved on STN
AN   2009041276  EMBASE
TI   Characterization of an alternative splice variant of LKB1.
AU   Denison, Fiona C.; Carling, David (correspondence); Woods, Angela
CS   Cellular Stress Group, Medical Research Council Clinical Sciences Centre,
     DuCane Road, London W12 0NN, United Kingdom. dcarling@imperial.ac.uk;
     angela.woods@imperial.ac.uk
AU   Hiscock, Natalie J.
CS   Unilever Discover, Personalised Vitality Platform, Colworth Science Park,
     Sharnbrook, Bedfordshire MK44 1LQ, United Kingdom.
SO   Journal of Biological Chemistry, (2 Jan 2009) Vol. 284, No. 1, pp. 67-76.
     Refs: 35
     ISSN: 0021-9258; E-ISSN: 1083-351X  CODEN: JBCHA3
PB   American Society for Biochemistry and Molecular Biology Inc., 9650
     Rockville Pike, Bethesda, MD 20814, United States.
CY   United States
DT   Journal; Article
FS   029     Clinical and Experimental Biochemistry
LA   English
SL   English
ED   Entered STN: 24 Feb 2009
     Last Updated on STN: 24 Feb 2009

L5   ANSWER 2 OF 15  HCAPLUS  COPYRIGHT 2010 ACS on STN
AN   2008:354110  HCAPLUS
DN   148:370238
TI   Insulin-resistant muscle is exercise resistant: evidence for reduced
     response of nuclear-encoded mitochondrial genes to exercise
AU   De Filippis, Elena; Alvarez, Guy; Berria, Rachele; Cusi, Kenneth; Everman,
     Sarah; Meyer, Christian; Mandarino, Lawrence J.
CS   Center for Metabolic Biology, Arizona State University, Tempe, AZ, USA
SO   American Journal of Physiology (2008), 294(3, Pt. 1), E607-E614
     CODEN: AJPHAP; ISSN: 0002-9513
PB   American Physiological Society
DT   Journal
LA   English
OSC.G   16     THERE ARE 16 CAPLUS RECORDS THAT CITE THIS RECORD (16 CITINGS)
RE.CNT  32     THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD
               ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5   ANSWER 3 OF 15  HCAPLUS  COPYRIGHT 2010 ACS on STN
AN   2008:1293529  HCAPLUS
DN   150:51992
TI   A novel short splice variant of the tumor suppressor LKB1 is required for
     spermiogenesis
AU   Towler, Mhairi C.; Fogarty, Sarah; Hawley, Simon A.; Pan, David A.;
     Martin, David M. A.; Morrice, Nicolas A.; McCarthy, Afshan; Galardo, Maria
     N.; Meroni, Silvina B.; Cigorraga, Selva B.; Ashworth, Alan; Sakamoto,
     Kei; Hardie, D. Grahame
```

CS Division of Molecular Physiology, School of Life Sciences, University of Dundee, Dundee, DD1 5EH, UK
SO Biochemical Journal (2008), 416(1), 1-14
CODEN: BIJOAK; ISSN: 0264-6021
PB Portland Press Ltd.
DT Journal
LA English
OSC.G 7 THERE ARE 7 CAPLUS RECORDS THAT CITE THIS RECORD (7 CITINGS)
RE.CNT 49 THERE ARE 49 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 4 OF 15 MEDLINE on STN DUPLICATE 1
AN 2007078449 MEDLINE
DN PubMed ID: 16985256
TI Effects of 3-phosphoglycerate and other metabolites on the activation of AMP-activated protein kinase by LKB1-STRAD-MO25.
AU Ellingson W J; Chesser D G; Winder W W
CS Department of Physiology and Developmental Biology, Brigham Young University, Provo, Utah 84602, USA.
SO American journal of physiology. Endocrinology and metabolism, (2007 Feb) Vol. 292, No. 2, pp. E400-7. Electronic Publication: 2006-09-19.
Journal code: 100901226. ISSN: 0193-1849. L-ISSN: 0193-1849.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200703
ED Entered STN: 7 Feb 2007
Last Updated on STN: 20 Mar 2007
Entered Medline: 19 Mar 2007

L5 ANSWER 5 OF 15 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN
AN 2007564307 EMBASE
TI Co-expression of LKB1, MO25α and STRADα in bacteria yield the functional and active heterotrimeric complex.
AU Neumann, Dietbert (correspondence); Suter, Marianne; Tuerk, Roland; Riek, Uwe; Wallimann, Theo
CS ETH Zurich, Institute of Cell Biology, HPM D23, Schafmattstr. 18, Zurich 8093, Switzerland. dietbert.neumann@cell.biol.ethz.ch
SO Molecular Biotechnology, (Jul 2007) Vol. 36, No. 3, pp. 220-231.
Refs: 25
ISSN: 1073-6085
CY United States
DT Journal; Article
FS 029 Clinical and Experimental Biochemistry
LA English
SL English
ED Entered STN: 4 Dec 2007
Last Updated on STN: 4 Dec 2007

L5 ANSWER 6 OF 15 Elsevier Biobase COPYRIGHT 2010 Elsevier Science B.V. on STN
AN 2007045887 ESBIOBASE
TI Effects of 3-phosphoglycerate and other metabolites on the activation of AMP-activated protein kinase by LKB1-STRAD-MO25
AU Ellingson, W.J.; Chesser, D.G.; Winder, W.W.
CS Ellingson, W.J.; Chesser, D.G.; Winder, W.W. (Department of Physiology and Developmental Biology, Brigham Young University, Provo, UT (US)); Winder, W.W. (545 WIDB, Brigham Young Univ., Provo, UT 84602 (US))
EMAIL: william_winder@byu.edu

SO American Journal of Physiology - Endocrinology and Metabolism (Feb 2007) Volume 292, Number 2, 47 refs.
CODEN: AJPMD9 ISSN: 0193-1849 E-ISSN: 1522-1555
DOI: 10.1152/ajpendo.00322.2006
CY United States of America
DT Journal; Article
LA English
SL English
ED Entered STN: 3 Feb 2009
Last updated on STN: 3 Feb 2009

L5 ANSWER 7 OF 15 Elsevier Biobase COPYRIGHT 2010 Elsevier Science B.V. on STN
AN 2006211772 ESBIOBASE
TI LKB1-dependent signaling pathways
AU Alessi, Dario R.; Sakamoto, Kei; Bayascas, Jose R.
CS Alessi, Dario R.; Sakamoto, Kei; Bayascas, Jose R. (Protein Phosphorylation Unit, School of Life Sciences, University of Dundee, Dundee DD1 5EH (GB))
EMAIL: d.r.alessi@dundee.ac.uk; k.sakamoto@dundee.ac.uk; j.bayascas@dundee.ac.uk
SO Annual Review of Biochemistry (2006) Volume 75, pp. 137-163, 141 refs.
CODEN: ARBOAW ISSN: 0066-4154
DOI: 10.1146/annurev.biochem.75.103004.142702
CY United States of America
DT Book; General Review; (Book Series)
LA English
SL English
ED Entered STN: 3 Feb 2009
Last updated on STN: 3 Feb 2009

L5 ANSWER 8 OF 15 MEDLINE on STN DUPLICATE 2
AN 2006105532 MEDLINE
DN PubMed ID: 16396636
TI The ubiquitin-associated domain of AMPK-related kinases regulates conformation and LKB1-mediated phosphorylation and activation.
AU Jaleel Mahaboobi; Villa Fabrizio; Deak Maria; Toth Rachel; Prescott Alan R; Van Aalten Daan M F; Alessi Dario R
CS MRC Protein Phosphorylation Unit, MSI/WTB Complex, University of Dundee, Dow Street, Dundee DD1 5EH, Scotland, UK.. a.mahaboobi@dundee.ac.uk
NC (United Kingdom Wellcome Trust)
SO The Biochemical journal, (2006 Mar 15) Vol. 394, No. Pt 3, pp. 545-55.
Journal code: 2984726R. E-ISSN: 1470-8728. L-ISSN: 0264-6021.
Report No.: NLM-PMC1383704.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals
EM 200607
ED Entered STN: 23 Feb 2006
Last Updated on STN: 29 Jul 2006
Entered Medline: 28 Jul 2006

L5 ANSWER 9 OF 15 MEDLINE on STN DUPLICATE 3
AN 2006342946 MEDLINE
DN PubMed ID: 16756488
TI LKB1-dependent signaling pathways.
AU Alessi Dario R; Sakamoto Kei; Bayascas Jose R
CS Medical Research Council, Protein Phosphorylation Unit, School of Life Sciences, University of Dundee, Dundee DD1 5EH, Scotland..

d.r.alessi@dundee.ac.uk
SO Annual review of biochemistry, (2006) Vol. 75, pp. 137-63. Ref: 139
Journal code: 2985150R. ISSN: 0066-4154. L-ISSN: 0066-4154.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
General Review; (REVIEW)
LA English
FS Priority Journals
EM 200702
ED Entered STN: 8 Jun 2006
Last Updated on STN: 6 Feb 2007
Entered Medline: 5 Feb 2007

L5 ANSWER 10 OF 15 WPIDS COPYRIGHT 2010 THOMSON REUTERS on STN
AN 2005-123150 [200513] WPIDS
DNC C2005-040924 [200513]
TI Identifying a modulator of AMPK (AMP-activated protein kinase) or AMPK subfamily member activation/phosphorylation in cell, useful for treating e.g. obesity, by determining if a test compound modulates LKB1 protein kinase activity
DC B04; D16
IN ALESSI D; BOUDEAU J; HARDIE D G; HARDIE D
PA (UYDU-N) UNIV DUNDEE; (ALES-I) ALESSI D; (BOUD-I) BOUDEAU J; (HARD-I) HARDIE D G
CYC 107
PIA WO 2005010174 A2 20050203 (200513)* EN 208[30]
EP 1651673 A2 20060503 (200629) EN
US 20070036793 A1 20070215 (200715) EN
JP 2007530000 W 20071101 (200780) JA 110
EP 1651673 B1 20080416 (200829) EN
DE 602004013160 E 20080529 (200838) DE
ES 2308198 T3 20081201 (200901) ES
DE 602004013160 T2 20090702 (200943) DE
ADT WO 2005010174 A2 WO 2004-GB3096 20040716; DE 602004013160 E DE 2004-602004013160 20040716; EP 1651673 A2 EP 2004-743435 20040716; EP 1651673 B1 EP 2004-743435 20040716; DE 602004013160 E EP 2004-743435 20040716; ES 2308198 T3 EP 2004-743435 20040716; EP 1651673 A2 PCT Application WO 2004-GB3096 20040716; US 20070036793 A1 PCT Application WO 2004-GB3096 20040716; JP 2007530000 W PCT Application WO 2004-GB3096 20040716; EP 1651673 B1 PCT Application WO 2004-GB3096 20040716; DE 602004013160 E PCT Application WO 2004-GB3096 20040716; JP 2007530000 W JP 2006-520011 20040716; US 20070036793 A1 US 2006-565058 20060621; DE 602004013160 T2 DE 2004-602004013160 20040716; DE 602004013160 T2 EP 2004-743435 20040716; DE 602004013160 T2 PCT Application WO 2004-GB3096 20040716
FDT DE 602004013160 E Based on EP 1651673 A; ES 2308198 T3 Based on EP 1651673 A; EP 1651673 A2 Based on WO 2005010174 A; JP 2007530000 W Based on WO 2005010174 A; EP 1651673 B1 Based on WO 2005010174 A; DE 602004013160 E Based on WO 2005010174 A; DE 602004013160 T2 Based on EP 1651673 A; DE 602004013160 T2 Based on WO 2005010174 A
PRAI GB 2003-30078 20031220
GB 2003-16725 20030717

L5 ANSWER 11 OF 15 MEDLINE on STN DUPLICATE 4
AN 2005600805 MEDLINE
DN PubMed ID: 16014350
TI Endurance training increases skeletal muscle LKB1 and PGC-1alpha protein abundance: effects of time and intensity.
AU Taylor Eric B; Lamb Jeremy D; Hurst Richard W; Chesser David G; Ellingson

William J; Greenwood Lyle J; Porter Brian B; Herway Seth T; Winder William W
CS Department of Physiology and Developmental Biology, 545 WIDB, Brigham Young University, Provo, UT 84602, USA.
NC AR 41438 (United States NIAMS NIH HHS)
SO American journal of physiology. Endocrinology and metabolism, (2005 Dec) Vol. 289, No. 6, pp. E960-8. Electronic Publication: 2005-07-12. Journal code: 100901226. ISSN: 0193-1849. L-ISSN: 0193-1849.
CY United States
DT (COMPARATIVE STUDY)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
LA English
FS Priority Journals
EM 200512
ED Entered STN: 11 Nov 2005
Last Updated on STN: 22 Dec 2005
Entered Medline: 21 Dec 2005

L5 ANSWER 12 OF 15 Elsevier Biobase COPYRIGHT 2010 Elsevier Science B.V. on STN
AN 2006123462 ESBIOBASE
TI Endurance training increases skeletal muscle LKB1 and PGC-1α protein abundance: Effects of time and intensity
AU Taylor, Eric B.; Lamb, Jeremy D.; Hurst, Richard W.; Chesser, David G.; Ellingson, William J.; Greenwood, Lyle J.; Porter, Brian B.; Herway, Seth T.; Winder, William W.
CS Taylor, Eric B.; Lamb, Jeremy D.; Hurst, Richard W.; Chesser, David G.; Ellingson, William J.; Greenwood, Lyle J.; Porter, Brian B.; Herway, Seth T.; Winder, William W. (Department of Physiology and Developmental Biology, Brigham Young University, Provo, UT (US)); Winder, William W. (545 WIDB, Brigham Young University, Provo, UT 84602 (US))
EMAIL: william_winder@byu.edu
SO American Journal of Physiology - Endocrinology and Metabolism (Dec 2005) Volume 289, Number 6, 68 refs.
CODEN: AJPMD9 ISSN: 0193-1849 E-ISSN: 1522-1555
DOI: 10.1152/ajpendo.00237.2005
CY United States of America
DT Journal; Article
LA English
SL English
ED Entered STN: 3 Feb 2009
Last updated on STN: 3 Feb 2009

L5 ANSWER 13 OF 15 HCAPLUS COPYRIGHT 2010 ACS on STN
AN 2005:113852 HCAPLUS
DN 142:174293
TI Analysis of the LKB1-STRAD-MO25 complex
AU Boudeau, Jerome; Scott, John W.; Resta, Nicoletta; Deak, Maria; Kieloch, Agnieszka; Komander, David; Hardie, D. Grahame; Prescott, Alan R.; Van Aalten, Daan M. F.; Alessi, Dario R.
CS MRC Protein Phosphorylation Unit, University of Dundee, Dundee, DD1 5EH, UK
SO Journal of Cell Science (2004), 117(26), 6365-6375
CODEN: JNCSAI; ISSN: 0021-9533
PB Company of Biologists Ltd.
DT Journal
LA English
OSC.G 44 THERE ARE 44 CAPLUS RECORDS THAT CITE THIS RECORD (44 CITINGS)
RE.CNT 51 THERE ARE 51 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

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L5   ANSWER 14 OF 15     MEDLINE on STN                DUPLICATE 5
AN   2004048988     MEDLINE
DN   PubMed ID: 14730349
TI   Crystal structure of MO25 alpha in complex with the C terminus
     of the pseudo kinase STE20-related adaptor.
AU   Milburn Christine C; Boudeau Jerome; Deak Maria; Alessi Dario R; van
     Aalten Daan M F
CS   Division of Biological Chemistry & Molecular Microbiology, School of Life
     Sciences, University of Dundee, Dundee DD1 5EH, Scotland.
SO   Nature structural & molecular biology, (2004 Feb) Vol. 11, No. 2, pp.
     193-200.  Electronic Publication: 2004-01-18.
     Journal code: 101186374. ISSN: 1545-9993. L-ISSN: 1545-9985.
CY   United States
DT   Journal; Article; (JOURNAL ARTICLE)
     (RESEARCH SUPPORT, NON-U.S. GOV'T)
LA   English
FS   Priority Journals
OS   PDB-1UPK; PDB-1UPL
EM   200404
ED   Entered STN: 30 Jan 2004
     Last Updated on STN: 6 Apr 2004
     Entered Medline: 5 Apr 2004

L5     ANSWER 15 OF 15 Elsevier Biobase COPYRIGHT 2010 Elsevier Science B.V. on
       STN
AN     2004032258   ESBIOBASE
TI     Crystal structure of MO25α in complex with the C
       terminus of the pseudo kinase STE20-related adaptor
AU     Milburn, Christine C.; Van Aalten, Daan M. F.; Boudeau, Jerome; Deak,
       Maria; Alessi, Dario R.
CS     Milburn, Christine C.; Van Aalten, Daan M. F. (Div. Biol. Chem./Molec.
       Microbiol., School of Life Sciences, University of Dundee, Dundee DD1
       5EH (GB)); Boudeau, Jerome; Deak, Maria; Alessi, Dario R. (MRC Protein
       Phosphorylation Unit, School of Life Sciences, University of Dundee,
       Dundee DD1 5EH (GB))
       EMAIL: dava@davapcl.bioch.dundee.ac.uk
SO     Nature Structural and Molecular Biology (Feb 2004) Volume 11, Number 2,
       pp. 193-200, 53 refs.
       CODEN: NSMBCU  ISSN: 1545-9993
       DOI: 10.1038/nsmb716
CY     United States of America
DT     Journal; Article
LA     English
SL     English
ED     Entered STN: 2 Feb 2009
       Last updated on STN: 2 Feb 2009


=> s mo25 (6A) mouse
L6          52 MO25 (6A) MOUSE

=>  s mo25 (4A) mouse
L7          52 MO25 (4A) MOUSE

=>  s mo25 (6A) human
L8          28 MO25 (6A) HUMAN

=>  s mo25 (4A) human
L9          28 MO25 (4A) HUMAN
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ENTER L# LIST OR (END):19
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WPIDS'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L9
L10          15 DUPLICATE REMOVE L9 (13 DUPLICATES REMOVED)

=> d l10 1-5 bib

L10  ANSWER 1 OF 15     MEDLINE on STN
AN   2010198307     MEDLINE
DN   PubMed ID: 20227367
TI   MicroRNA-451 regulates LKB1/AMPK signaling and allows adaptation to
     metabolic stress in glioma cells.
AU   Godlewski Jakub; Nowicki Michal O; Bronisz Agnieszka; Nuovo Gerard;
     Palatini Jeff; De Lay Michael; Van Brocklyn James; Ostrowski Michael C;
     Chiocca E Antonio; Lawler Sean E
CS   Dardinger Laboratory for Neuro-oncology and Neurosciences, Department of
     Neurological Surgery, The Ohio State University Medical Center and James
     Comprehensive Cancer Center, Columbus, OH 43210, USA.
SO   Molecular cell, (2010 Mar 12) Vol. 37, No. 5, pp. 620-32.
     Journal code: 9802571. E-ISSN: 1097-4164. L-ISSN: 1097-2765.
CY   United States
DT   Journal; Article; (JOURNAL ARTICLE)
     (RESEARCH SUPPORT, NON-U.S. GOV'T)
LA   English
FS   Priority Journals
EM   201004
ED   Entered STN: 23 Mar 2010
     Last Updated on STN: 14 Apr 2010
     Entered Medline: 13 Apr 2010

L10  ANSWER 2 OF 15     MEDLINE on STN                  DUPLICATE 1
AN   2010153017     MEDLINE
DN   PubMed ID: 20197543
TI   Allosteric protein kinase regulation by pseudokinases: insights from
     STRAD.
AU   Rajakulendran Thanashan; Sicheri Frank
CS   1Centre for Systems Biology, Samuel Lunenfeld Research Institute, Toronto,
     Ontario M5G 1X5, Canada.
SO   Science signaling, (2010) Vol. 3, No. 111, pp. pe8.  Electronic
     Publication: 2010-03-02. Ref: 15
     Journal code: 101465400. E-ISSN: 1937-9145.
CY   United States
DT   Journal; Article; (JOURNAL ARTICLE)
     General Review; (REVIEW)
LA   English
FS   Priority Journals
EM   201005
ED   Entered STN: 4 Mar 2010
     Last Updated on STN: 26 May 2010
     Entered Medline: 25 May 2010

L10  ANSWER 3 OF 15     MEDLINE on STN
AN   2009829133     MEDLINE
DN   PubMed ID: 19892943
TI   Structure of the LKB1-STRAD-MO25 complex reveals an allosteric mechanism
     of kinase activation.
AU   Zeqiraj Elton; Filippi Beatrice Maria; Deak Maria; Alessi Dario R; van
```

Aalten Daan M F
CS Division of Molecular Microbiology, College of Life Sciences, University of Dundee, Dundee DD1 5EH, Scotland.
NC C33794/A10969 (United Kingdom Cancer Research UK)
(United Kingdom Wellcome Trust)
SO Science (New York, N.Y.), (2009 Dec 18) Vol. 326, No. 5960, pp. 1707-11. Electronic Publication: 2009-11-05.
Journal code: 0404511. E-ISSN: 1095-9203. L-ISSN: 0036-8075.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals
OS PDB-2WTK
EM 201001
ED Entered STN: 19 Dec 2009
Last Updated on STN: 26 Jan 2010
Entered Medline: 21 Jan 2010

L10 ANSWER 4 OF 15 MEDLINE on STN DUPLICATE 2
AN 2009295322 MEDLINE
DN PubMed ID: 19386264
TI Mst4 and Ezrin induce brush borders downstream of the Lkb1/Strad/Mo25 polarization complex.
AU ten Klooster Jean Paul; Jansen Marnix; Yuan Jin; Oorschot Viola; Begthel Harry; Di Giacomo Valeria; Colland Frederic; de Koning John; Maurice Madelon M; Hornbeck Peter; Clevers Hans
CS Hubrecht Institute, KNAW and University Medical Centre, Utrecht, The Netherlands.
SO Developmental cell, (2009 Apr) Vol. 16, No. 4, pp. 551-62.
Journal code: 101120028. E-ISSN: 1878-1551. L-ISSN: 1534-5807.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals
EM 200905
ED Entered STN: 24 Apr 2009
Last Updated on STN: 16 May 2009
Entered Medline: 15 May 2009

L10 ANSWER 5 OF 15 MEDLINE on STN DUPLICATE 3
AN 2009406809 MEDLINE
DN PubMed ID: 19513107
TI ATP and MO25alpha regulate the conformational state of the STRADalpha pseudokinase and activation of the LKB1 tumour suppressor.
AU Zeqiraj Elton; Filippi Beatrice Maria; Goldie Simon; Navratilova Iva; Boudeau Jerome; Deak Maria; Alessi Dario R; van Aalten Daan M F
CS Division of Molecular Microbiology, College of Life Sciences, University of Dundee, Dundee, Scotland.
NC (United Kingdom Medical Research Council)
(United Kingdom Wellcome Trust)
SO PLoS biology, (2009 Jun 9) Vol. 7, No. 6, pp. e1000126. Electronic Publication: 2009-06-09.
Journal code: 101183755. E-ISSN: 1545-7885. L-ISSN: 1544-9173.
Report No.: NLM-PMC2686265.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals

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EM   200908
ED   Entered STN: 11 Jun 2009
     Last Updated on STN: 1 Sep 2009
     Entered Medline: 31 Aug 2009


=>

=> d l10 15 bib

L10  ANSWER 15 OF 15  WPIDS COPYRIGHT 2010       THOMSON REUTERS on STN
AN   2001-308142 [200132]   WPIDS
DNC  C2001-095180 [200132]
TI   Novel human acute neuronal induced calcium binding polypeptide, and
     polynucleotides encoding them useful for diagnosing or treating stroke,
     acute head trauma, multiple sclerosis and spinal cord injury
DC   B04; D16
IN   DEN DAAS I; DUECKER K
PA   (MERE-C) MERCK PATENT GMBH
CYC  28
PIA  WO 2001023552   A1 20010405 (200132)* EN  45[1]
     EP 1214413      A1 20020619 (200240)  EN
     JP 2003510076   W  20030318 (200321)  JA  51
ADT  WO 2001023552 A1 WO 2000-EP9132 20000918; EP 1214413 A1 EP 2000-967699
     20000918; EP 1214413 A1 WO 2000-EP9132 20000918; JP 2003510076 W WO
     2000-EP9132 20000918; JP 2003510076 W JP 2001-526934 20000918
FDT  EP 1214413 A1 Based on WO 2001023552 A; JP 2003510076 W Based on WO
     2001023552 A
PRAI EP 1999-118848       19990924


=> d l10 10-15 bib ab

L10  ANSWER 10 OF 15     MEDLINE on STN                 DUPLICATE 4
AN   2005644516     MEDLINE
DN   PubMed ID: 16325501
TI   The fission yeast MO25 protein functions in polar growth and cell
     separation.
AU   Mendoza Manuel; Redemann Stefanie; Brunner Damian
CS   European Molecular Biology Laboratory, Heidelberg, Germany.
SO   European journal of cell biology, (2005 Dec) Vol. 84, No. 12, pp. 915-26.
     Electronic Publication: 2005-10-03.
     Journal code: 7906240. ISSN: 0171-9335. L-ISSN: 0171-9335.
CY   Germany: Germany, Federal Republic of
DT   Journal; Article; (JOURNAL ARTICLE)
     (RESEARCH SUPPORT, NON-U.S. GOV'T)
LA   English
FS   Priority Journals
EM   200603
ED   Entered STN: 6 Dec 2005
     Last Updated on STN: 3 Mar 2006
     Entered Medline: 2 Mar 2006
AB   Proteins of the MO25 family are widely conserved but their function has
     not been characterized in detail.  Human MO25 is a
     cofactor of LKB1, a conserved protein kinase with roles in cell polarity
     in nematodes, flies and mammalian cells.  Furthermore, the budding yeast
     MO25 homologue, Hym1, is important for cell separation and morphogenesis.
     We have characterized Pmo25p, the MO25 homologue in the fission yeast
     Schizosaccharomyces pombe.  Pmo25p is an essential protein required for
     polar growth; in its absence the actin cytoskeleton becomes depolarized
     and cells adopt a round morphology.  In addition, pmo25 mutants are
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defective in cell separation. Both functions of Pmo25p appear to be mediated by the Orb6p-Mob2p kinase complex. Pmo25p shows no distinct localization during interphase, but it is recruited to one of the two spindle pole bodies during anaphase and to the division site during cytokinesis. The septation initiation network (SIN) regulates the localization of Pmo25p, suggesting that it regulates Pmo25p function during cell division.

L10 ANSWER 11 OF 15 HCAPLUS COPYRIGHT 2010 ACS on STN
AN 2004:239364 HCAPLUS
DN 140:419521
TI Comprehensive Proteomic Analysis of Human Par Protein Complexes Reveals an Interconnected Protein Network
AU Brajenovic, Miro; Joberty, Gerard; Kuester, Bernhard; Bouwmeester, Tewis; Drewes, Gerard
CS Cellzome AG, Heidelberg, D-69117, Germany
SO Journal of Biological Chemistry (2004), 279(13), 12804-12811
CODEN: JBCHA3; ISSN: 0021-9258
PB American Society for Biochemistry and Molecular Biology
DT Journal
LA English
AB The polarization of eukaryotic cells is controlled by the concerted activities of asym. localized proteins. The PAR proteins, first identified in Caenorhabditis elegans, are common regulators of cell polarity conserved from nematode and flies to man. However, little is known about the mol. mechanisms by which these proteins and protein complexes establish cell polarity in mammals. We have mapped multiprotein complexes formed around the putative human Par orthologs MARK4 (microtubule-associated protein/microtubule affinity-regulating kinase 4) (Par-1), Par-3, LKB1 (Par-4), 14-3-3ζ and η (Par-5), Par-6a, -b, -c, and PKCλ (PKC3). We employed a proteomic approach comprising tandem affinity purification (TAP) of protein complexes from cultured cells and protein sequencing by tandem mass spectrometry. From these data we constructed a highly interconnected protein network consisting of three core complex "modules" formed around MARK4 (Par-1), Par-3·Par-6, and LKB1 (Par-4). The network confirms most previously reported interactions. In addition we identified more than 50 novel interactors, some of which, like the 14-3-3 phospho-protein scaffolds, occur in more than one distinct complex. We demonstrate that the complex formation between LKB1·Par-4, PAPK, and Mo25 results in the translocation of LKB1 from the nucleus to the cytoplasm and to tight junctions and show that the LKB1 complex may activate MARKs, which are known to introduce 14-3-3 binding sites into several substrates. Our findings suggest co-regulation and/or signaling events between the distinct Par complexes and provide a basis for further elucidation of the mol. mechanisms that govern cell polarity.
OSC.G 72 THERE ARE 72 CAPLUS RECORDS THAT CITE THIS RECORD (72 CITINGS)
RE.CNT 58 THERE ARE 58 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 12 OF 15 HCAPLUS COPYRIGHT 2010 ACS on STN
AN 2005:113852 HCAPLUS
DN 142:174293
TI Analysis of the LKB1-STRAD-MO25 complex
AU Boudeau, Jerome; Scott, John W.; Resta, Nicoletta; Deak, Maria; Kieloch, Agnieszka; Komander, David; Hardie, D. Grahame; Prescott, Alan R.; Van Aalten, Daan M. F.; Alessi, Dario R.
CS MRC Protein Phosphorylation Unit, University of Dundee, Dundee, DD1 5EH, UK
SO Journal of Cell Science (2004), 117(26), 6365-6375
CODEN: JNCSAI; ISSN: 0021-9533

PB Company of Biologists Ltd.
DT Journal
LA English
AB Mutations in the LKB1 tumor suppressor threonine kinase cause the inherited Peutz-Jeghers cancer syndrome and are also observed in some sporadic cancers. Recent work indicates that LKB1 exerts effects on metabolism, polarity and proliferation by phosphorylating and activating protein kinases belonging to the AMPK subfamily. In vivo, LKB1 forms a complex with STRAD, an inactive pseudo-kinase, and MO25, an armadillo repeat scaffolding-like protein. Binding of LKB1 to STRAD-MO25 activates LKB1 and re-localizes it from the nucleus to the cytoplasm. To learn more about the inherent properties of the LKB1-STRAD-MO25 complex, we first investigated the activity of 34 point mutants of LKB1 found in human cancers and their ability to interact with STRAD and MO25. Interestingly, 12 of these mutants failed to interact with STRAD-MO25. Performing mutagenesis anal., we defined two binding sites located on opposite surfaces of MO25α, which are required for the assembly of MO25α into a complex with STRADα and LKB1. In addition, we demonstrate that LKB1 does not require phosphorylation of its own T-loop to be activated by STRADα-MO25α, and discuss the possibility that this unusual mechanism of regulation arises from LKB1 functioning as an upstream kinase. Finally, we establish that STRADα, despite being catalytically inactive, is still capable of binding ATP with high affinity, but that this is not required for activation of LKB1. Taken together, our findings reinforce the functional importance of the binding of LKB1 to STRAD, and provide a greater understanding of the mechanism by which LKB1 is regulated and activated through its interaction with STRAD and MO25.
OSC.G 44 THERE ARE 44 CAPLUS RECORDS THAT CITE THIS RECORD (44 CITINGS)
RE.CNT 51 THERE ARE 51 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 13 OF 15 HCAPLUS COPYRIGHT 2010 ACS on STN
AN 2004:106951 HCAPLUS
DN 140:316769
TI Crystal structure of MO25α in complex with the C terminus of the pseudo kinase STE20-related adaptor
AU Milburn, Christine C.; Boudeau, Jerome; Deak, Maria; Alessi, Dario R.; van Aalten, Daan M. F.
CS School of Life Sciences, Division of Biological Chemistry & Molecular Microbiology, University of Dundee, Dundee, DD1 5EH, UK
SO Nature Structural & Molecular Biology (2004), 11(2), 193-200
CODEN: NSMBCU; ISSN: 1545-9993
PB Nature Publishing Group
DT Journal
LA English
AB Mouse protein 25α (MO25α) is a 40-kDa protein that together with the STE20-related adaptor-α (STRADα) pseudokinase, forms a regulatory complex capable of stimulating the activity of the LKB1 tumor suppressor protein kinase. The latter is mutated in the inherited Peutz-Jeghers cancer syndrome (PJS). MO25α binds directly to a conserved Trp-Glu-Phe sequence at the STRADα C terminus, markedly enhancing binding of STRADα to LKB1 and increasing LKB1 catalytic activity. The MO25α crystal structure reveals a helical repeat fold, distantly related to the Armadillo proteins. A complex with the STRADα peptide reveals a hydrophobic pocket that is involved in a unique and specific interaction with the Trp-Glu-Phe motif, further supported by mutagenesis studies. The data represent a first step toward structural anal. of the LKB1-STRAD-MO25 complex, and suggests that MO25α is a scaffold protein to which other regions of STRAD-LKB1, cellular LKB1 substrates or regulatory components could bind.

OSC.G 33 THERE ARE 33 CAPLUS RECORDS THAT CITE THIS RECORD (33 CITINGS)
RE.CNT 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 14 OF 15 MEDLINE on STN DUPLICATE 5
AN 2003489699 MEDLINE
DN PubMed ID: 14517248
TI MO25alpha/beta interact with STRADalpha/beta enhancing their ability to bind, activate and localize LKB1 in the cytoplasm.
AU Boudeau Jerome; Baas Annette F; Deak Maria; Morrice Nick A; Kieloch Agnieszka; Schutkowski Mike; Prescott Alan R; Clevers Hans C; Alessi Dario R
CS MRC Protein Phosphorylation Unit, School of Life Sciences, MSI/WTB Complex, University of Dundee, Dow Street, Dundee DD1 5EH, UK.. j.boudeau@dundee.ac.uk
SO The EMBO journal, (2003 Oct 1) Vol. 22, No. 19, pp. 5102-14. Journal code: 8208664. ISSN: 0261-4189. L-ISSN: 0261-4189. Report No.: NLM-PMC204473.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals
EM 200311
ED Entered STN: 22 Oct 2003
Last Updated on STN: 19 Dec 2003
Entered Medline: 24 Nov 2003
AB Mutations in the LKB1 protein kinase result in the inherited Peutz Jeghers cancer syndrome. LKB1 has been implicated in regulating cell proliferation and polarity although little is known about how this enzyme is regulated. We recently showed that LKB1 is activated through its interaction with STRADalpha, a catalytically deficient pseudokinase. Here we show that endogenous LKB1-STRADalpha complex is associated with a protein of unknown function, termed MO25alpha, through the interaction of MO25alpha with the last three residues of STRADalpha. MO25alpha and STRADalpha anchor LKB1 in the cytoplasm, excluding it from the nucleus. Moreover, MO25alpha enhances the formation of the LKB1-STRADalpha complex in vivo, stimulating the catalytic activity of LKB1 approximately 10-fold. We demonstrate that the related STRADbeta and MO25beta isoforms are also able to stabilize LKB1 in an active complex and that it is possible to isolate complexes of LKB1 bound to STRAD and MO25 isoforms, in which the subunits are present in equimolar amounts. Our results indicate that MO25 may function as a scaffolding component of the LKB1-STRAD complex and plays a crucial role in regulating LKB1 activity and cellular localization.

L10 ANSWER 15 OF 15 WPIDS COPYRIGHT 2010 THOMSON REUTERS on STN
AN 2001-308142 [200132] WPIDS
DNC C2001-095180 [200132]
TI Novel human acute neuronal induced calcium binding polypeptide, and polynucleotides encoding them useful for diagnosing or treating stroke, acute head trauma, multiple sclerosis and spinal cord injury
DC B04; D16
IN DEN DAAS I; DUECKER K
PA (MERE-C) MERCK PATENT GMBH
CYC 28
PIA WO 2001023552 A1 20010405 (200132)* EN 45[1]
EP 1214413 A1 20020619 (200240) EN
JP 2003510076 W 20030318 (200321) JA 51
ADT WO 2001023552 A1 WO 2000-EP9132 20000918; EP 1214413 A1 EP 2000-967699 20000918; EP 1214413 A1 WO 2000-EP9132 20000918; JP 2003510076 W WO

2000-EP9132 20000918; JP 2003510076 W JP 2001-526934 20000918
FDT EP 1214413 A1 Based on WO 2001023552 A; JP 2003510076 W Based on WO 2001023552 A
PRAI EP 1999-118848 19990924
AB WO 2001023552 A1 UPAB: 20050525
NOVELTY - Human acute neuronal induced calcium binding protein (ANIC-BP) (I), is new.

DETAILED DESCRIPTION - (I) is an isolated polypeptide:

(i) encoded by a polynucleotide comprising a fully defined sequence of 1014 nucleotides (S1) as given in the specification;

(ii) comprising a polypeptide sequence having 95% identity to a fully defined sequence of 337 amino acids (S2) as given in the specification;

(iii) having 95% identity to (S2); or

(iv) (S2) or (v) fragments or variants of the above mentioned polypeptides

INDEPENDENT CLAIMS are also included for the following:

(1) an isolated polynucleotide (II) selected from a group which comprises:

(i) polynucleotide sequence having at least 95% identity to the polynucleotide sequence of (S1);

(ii) comprising a polynucleotide sequence encoding a polypeptide sequence having at least 95% identity to the polypeptide sequence of (S2);

(iii) with a nucleotide sequence of at least 100 nucleotides obtained by screening a library under stringent hybridization conditions with a labeled probe having the sequence of (S1) or its fragment having at least 15 nucleotides;

(iv) the RNA equivalent of the (i) or (ii); or

(v) a polynucleotide sequence complementary to (i-iv) or polynucleotides that are variants and fragments to (i-iv) or complementary to (i-iv);

(2) an expression system (III) comprising (II) which is capable of producing (I) when present in a compatible host cell;

(3) a recombinant host cell (IV), comprising (III) or its membrane, expressing (I);

(4) preparation of (I);

(5) a fusion protein comprising immunoglobulin Fc-region and (I);

(6) an antibody (V) specific for (I);

(7) screening to identify compounds that stimulate or that inhibit the function of (I) involves measuring or detecting, qualitatively or quantitatively, the binding of the candidate compound to (I) (or to the cells or membranes bearing the polypeptide) or a fusion protein by means of a label directly or indirectly associated with the candidate compound;

(a) measuring the competition of binding of the candidate compound to the polypeptide or its fusion protein in the presence of a labeled competitor;

(b) testing whether the candidate compounds results in a signal generated by activation or inhibition of the polypeptide using appropriate detection systems;

(c) mixing a candidate compound with a solution comprising (I) to form a mixture, measuring the activity of the polypeptide in the mixture and comparing the activity of the mixture to a control mixture which contains no candidate compound; or

(d) detecting the effect of the candidate compound on the production of mRNA encoding the polypeptide or the polypeptide in cells, using an enzyme linked immunosorbent assay (ELISA) and producing the compound according to standard biotechnological or chemical technique.

ACTIVITY - Cerebroprotective; neuroprotective; vulnerary. No supporting data is given.

MECHANISM OF ACTION - Vaccine; gene therapy.

USE - (I), (II) are useful for treating stroke, acute head trauma,

multiple sclerosis and spinal cord injury. (I), (II) are also useful as vaccines for inducing an immunological response in a mammal. Fragments and variants of (I) are useful for producing the corresponding full length polypeptide by peptide synthesis. (I) is also used to identify membrane bound or soluble receptors. Polynucleotides that are identical or have sufficient identity to (II) having a sequence of (S1) may be used as hybridization probes for cDNA and genomic DNA or as primers for a nucleic acid amplification reaction. The probes and primers may be used to isolate cDNA and genomic clones of other genes that have a sequence similarity to (S1). (II) may also be used as a diagnostic reagent through detection of mutations in the associated gene. Detection of a mutated form of the gene characterized by the polynucleotide of (S1) in the cDNA or genomic sequence and which is associated with the dysfunction will provide a diagnostic tool to diagnose a disease or susceptibility to a disease resulting from underexpression, overexpression or altered spatial or temporal expression of the gene. (II) is also valuable for chromosome localization studies, tissue expression studies. Detection of abnormally decreased or increased levels of (I) or mRNA expression may also be used for diagnosing or determining susceptibility of a subject to a disease. (I), (II), (V) are used to configure screening methods for detecting the effect of added compounds on the production of mRNA and polypeptide in the cells.

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=> duplicate
ENTER REMOVE, IDENTIFY, ONLY, OR (?):remove
ENTER L# LIST OR (END):l7
DUPLICATE PREFERENCE IS 'MEDLINE, SCISEARCH, LIFESCI, BIOSIS, EMBASE, HCAPLUS,
ESBIOBASE, BIOTECHNO, WPIDS, BIOENG'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L7
L11          16 DUPLICATE REMOVE L7 (36 DUPLICATES REMOVED)

=> d l11 10-16 bib ab

L11    ANSWER 10 OF 16 Elsevier Biobase COPYRIGHT 2010 Elsevier Science B.V. on
       STN
AN     2006123462   ESBIOBASE
TI     Endurance training increases skeletal muscle LKB1 and PGC-1α
       protein abundance: Effects of time and intensity
AU     Taylor, Eric B.; Lamb, Jeremy D.; Hurst, Richard W.; Chesser, David G.;
       Ellingson, William J.; Greenwood, Lyle J.; Porter, Brian B.; Herway,
       Seth T.; Winder, William W.
CS     Taylor, Eric B.; Lamb, Jeremy D.; Hurst, Richard W.; Chesser, David G.;
       Ellingson, William J.; Greenwood, Lyle J.; Porter, Brian B.; Herway,
       Seth T.; Winder, William W. (Department of Physiology and Developmental
       Biology, Brigham Young University, Provo, UT (US)); Winder, William W.
       (545 WIDB, Brigham Young University, Provo, UT 84602 (US))
       EMAIL: william_winder@byu.edu
SO     American Journal of Physiology - Endocrinology and Metabolism (Dec 2005)
       Volume 289, Number 6, 68 refs.
       CODEN: AJPMD9  ISSN: 0193-1849  E-ISSN: 1522-1555
       DOI: 10.1152/ajpendo.00237.2005
CY     United States of America
DT     Journal; Article
LA     English
SL     English
ED     Entered STN: 3 Feb 2009
       Last updated on STN: 3 Feb 2009
AB     Recent research suggests that LKB1 is the major AMP-activated protein
       kinase kinase (AMPKK). Peroxisome-proliferator-activated
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receptor-γ coactivator-1α (PGC-1α) is a master coordinator of mitochondrial biogenesis. Previously we reported that skeletal muscle LKB1 protein increases with endurance training. The purpose of this study was to determine whether training-induced increases in skeletal muscle LKB1 and PGC-1α protein exhibit a time course and intensity-dependent response similar to that of citrate synthase. Male Sprague-Dawley rats completed endurance- and interval-training protocols. For endurance training, rats trained for 4, 11, 25, or 53 days. Interval-training rats trained identically to endurance-trained rats, except that after 25 days interval training was combined with endurance training. Time course data were collected from endurance-trained red quadriceps (RQ) after each time point. Interval training data were collected from soleus, RQ, and white quadriceps (WQ) muscle after 53 days only. Mouse protein 25 (MO25) and PGC-1α protein increased significantly after 4 days. Increased citrate synthase activity, increased LKB1 protein, and decreased AMPKK activity were found after 11 days. Maximal increases occurred after 4 days for hexokinase II, 25 days for MO25, and 53 days for citrate synthase, LKB1, and PGC-1α. In WQ, but not RQ or soleus, interval training had an additive effect to endurance training and induced significant increases in all proteins measured. These results demonstrate that LKB1 and PGC-1α protein abundances increase with endurance and interval training similarly to citrate synthase. The increase in LKB1 and PGC-1α with endurance and interval training may function to maintain the training-induced increases in mitochondrial mass. Copyright .COPYRGT. 2005 the American Physiological Society.

L11 ANSWER 11 OF 16 HCAPLUS COPYRIGHT 2010 ACS on STN
AN 2005:113852 HCAPLUS
DN 142:174293
TI Analysis of the LKB1-STRAD-MO25 complex
AU Boudeau, Jerome; Scott, John W.; Resta, Nicoletta; Deak, Maria; Kieloch, Agnieszka; Komander, David; Hardie, D. Grahame; Prescott, Alan R.; Van Aalten, Daan M. F.; Alessi, Dario R.
CS MRC Protein Phosphorylation Unit, University of Dundee, Dundee, DD1 5EH, UK
SO Journal of Cell Science (2004), 117(26), 6365-6375
CODEN: JNCSAI; ISSN: 0021-9533
PB Company of Biologists Ltd.
DT Journal
LA English
AB Mutations in the LKB1 tumor suppressor threonine kinase cause the inherited Peutz-Jeghers cancer syndrome and are also observed in some sporadic cancers. Recent work indicates that LKB1 exerts effects on metabolism, polarity and proliferation by phosphorylating and activating protein kinases belonging to the AMPK subfamily. In vivo, LKB1 forms a complex with STRAD, an inactive pseudo-kinase, and MO25, an armadillo repeat scaffolding-like protein. Binding of LKB1 to STRAD-MO25 activates LKB1 and re-localizes it from the nucleus to the cytoplasm. To learn more about the inherent properties of the LKB1-STRAD-MO25 complex, we first investigated the activity of 34 point mutants of LKB1 found in human cancers and their ability to interact with STRAD and MO25. Interestingly, 12 of these mutants failed to interact with STRAD-MO25. Performing mutagenesis anal., we defined two binding sites located on opposite surfaces of MO25α, which are required for the assembly of MO25α into a complex with STRADα and LKB1. In addition, we demonstrate that LKB1 does not require phosphorylation of its own T-loop to be activated by STRADα-MO25α, and discuss the possibility that this unusual mechanism of regulation arises from LKB1 functioning as an upstream kinase. Finally, we establish that STRADα, despite being catalytically inactive, is still capable of binding ATP with high

affinity, but that this is not required for activation of LKB1. Taken together, our findings reinforce the functional importance of the binding of LKB1 to STRAD, and provide a greater understanding of the mechanism by which LKB1 is regulated and activated through its interaction with STRAD and MO25.

OSC.G 44 THERE ARE 44 CAPLUS RECORDS THAT CITE THIS RECORD (44 CITINGS)
RE.CNT 51 THERE ARE 51 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 12 OF 16 MEDLINE on STN DUPLICATE 5
AN 2004048988 MEDLINE
DN PubMed ID: 14730349
TI Crystal structure of MO25 alpha in complex with the C terminus of the pseudo kinase STE20-related adaptor.
AU Milburn Christine C; Boudeau Jerome; Deak Maria; Alessi Dario R; van Aalten Daan M F
CS Division of Biological Chemistry & Molecular Microbiology, School of Life Sciences, University of Dundee, Dundee DD1 5EH, Scotland.
SO Nature structural & molecular biology, (2004 Feb) Vol. 11, No. 2, pp. 193-200. Electronic Publication: 2004-01-18.
Journal code: 101186374. ISSN: 1545-9993. L-ISSN: 1545-9985.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals
OS PDB-1UPK; PDB-1UPL
EM 200404
ED Entered STN: 30 Jan 2004
Last Updated on STN: 6 Apr 2004
Entered Medline: 5 Apr 2004
AB Mouse protein 25 alpha (MO25 alpha) is a 40-kDa protein that, together with the STE20-related adaptor-alpha (STRAD alpha) pseudo kinase, forms a regulatory complex capable of stimulating the activity of the LKB1 tumor suppressor protein kinase. The latter is mutated in the inherited Peutz-Jeghers cancer syndrome (PJS). MO25 alpha binds directly to a conserved Trp-Glu-Phe sequence at the STRAD alpha C terminus, markedly enhancing binding of STRAD alpha to LKB1 and increasing LKB1 catalytic activity. The MO25 alpha crystal structure reveals a helical repeat fold, distantly related to the Armadillo proteins. A complex with the STRAD alpha peptide reveals a hydrophobic pocket that is involved in a unique and specific interaction with the Trp-Glu-Phe motif, further supported by mutagenesis studies. The data represent a first step toward structural analysis of the LKB1-STRAD-MO25 complex, and suggests that MO25 alpha is a scaffold protein to which other regions of STRAD-LKB1, cellular LKB1 substrates or regulatory components could bind.

L11 ANSWER 13 OF 16 Elsevier Biobase COPYRIGHT 2010 Elsevier Science B.V. on STN
AN 2004032258 ESBIOBASE
TI Crystal structure of MO25α in complex with the C terminus of the pseudo kinase STE20-related adaptor
AU Milburn, Christine C.; Van Aalten, Daan M. F.; Boudeau, Jerome; Deak, Maria; Alessi, Dario R.
CS Milburn, Christine C.; Van Aalten, Daan M. F. (Div. Biol. Chem./Molec. Microbiol., School of Life Sciences, University of Dundee, Dundee DD1 5EH (GB)); Boudeau, Jerome; Deak, Maria; Alessi, Dario R. (MRC Protein Phosphorylation Unit, School of Life Sciences, University of Dundee, Dundee DD1 5EH (GB))
EMAIL: dava@davapc1.bioch.dundee.ac.uk
SO Nature Structural and Molecular Biology (Feb 2004) Volume 11, Number 2,

pp. 193-200, 53 refs.
CODEN: NSMBCU ISSN: 1545-9993
DOI: 10.1038/nsmb716
CY United States of America
DT Journal; Article
LA English
SL English
ED Entered STN: 2 Feb 2009
Last updated on STN: 2 Feb 2009
AB Mouse protein 25α (MO25α) is a 40-kDa protein that, together with the STE20-related adaptor-α (STRADα) pseudo kinase, forms a regulatory complex capable of stimulating the activity of the LKB1 tumor suppressor protein kinase. The latter is mutated in the inherited Peutz-Jeghers cancer syndrome (PJS). MO25α binds directly to a conserved Trp-Glu-Phe sequence at the STRADα C terminus, markedly enhancing binding of STRADα to LKB1 and increasing LKB1 catalytic activity. The MO25α crystal structure reveals a helical repeat fold, distantly related to the Armadillo proteins. A complex with the STRADα peptide reveals a hydrophobic pocket that is involved in a unique and specific interaction with the Trp-Glu-Phe motif, further supported by mutagenesis studies. The data represent a first step toward structural analysis of the LKB1-STRAD-MO25 complex, and suggests that MO25α is a scaffold protein to which other regions of STRAD-LKB1, cellular LKB1 substrates or regulatory components could bind.

L11 ANSWER 14 OF 16 MEDLINE on STN DUPLICATE 6
AN 1999126010 MEDLINE
DN PubMed ID: 9928930
TI Molecular characterization of HymA, an evolutionarily highly conserved and highly expressed protein of Aspergillus nidulans.
AU Karos M; Fischer R
CS Laboratorium fur Mikrobiologie, Philipps-Universitat Marburg and Max-Planck-Institut fur terrestrische Mikrobiologie, Germany.
SO Molecular & general genetics : MGG, (1999 Jan) Vol. 260, No. 6, pp. 510-21.
Journal code: 0125036. ISSN: 0026-8925. L-ISSN: 0026-8925.
CY GERMANY: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals
OS GENBANK-AJ001157
EM 199902
ED Entered STN: 1 Mar 1999
Last Updated on STN: 4 Mar 2003
Entered Medline: 18 Feb 1999
AB Aspergillus nidulans reproduces asexually via uninucleate, haploid spores, which are produced on morphologically differentiated aerial structures, called conidiophores. These consist of four distinct cell types, a foot with a terminally swollen stalk, metulae, phialides and conidiospores. The molecular mechanisms underlying the morphological changes that occur during conidiophore development have been studied by mutant analysis. We have isolated the hym A mutant, in which conidiophore development is affected at the metula stage. In the mutant metulae do not differentiate properly but come to resemble hyphae (hym = hypha-like metulae). In this paper we have analyzed the corresponding gene. It encodes a highly expressed 44 kDa protein which resides in the cytoplasm and has homologues in yeast, plants, fly, worm, fish, mice and man. We constructed hym deletion strains of Saccharomyces cerevisiae and of A. nidulans and found that the gene is essential in S. cerevisiae but is dispensable in the

filamentous fungus. A cellular function for the Hym protein has not yet been defined in any organism. To demonstrate functional conservation we constructed a chimeric protein comprised of the N-terminal half of the A. nidulans and the C-terminal half of the mouse homologue MO25. This hybrid protein could fully substitute for HymA function in A. nidulans. In addition, the mouse protein itself partially rescued the hym A mutation in the fungus. HymA is thus highly conserved in evolution and probably serves similar functions. The fact that hym A is required for conidiophore development in A. nidulans suggests that homologous genes in other organisms might also be involved in morphogenesis.

L11 ANSWER 15 OF 16 HCAPLUS COPYRIGHT 2010 ACS on STN
AN 1999:112463 HCAPLUS
DN 130:321416
TI Molecular characterization of HymA, an evolutionarily highly conserved and highly expressed protein of Aspergillus nidulans
AU Karos, M.; Fischer, R.
CS Laboratorium fur Mikrobiologie Philipps-Universitat Marburg, Max-Planck-Institut fur terrestrische Mikrobiologie Karl-von-Frisch-Str., Marburg, D-35043, Germany
SO Molecular and General Genetics (1998), 260(6), 510-521
CODEN: MGGEAE; ISSN: 0026-8925
PB Springer-Verlag
DT Journal
LA English
AB Aspergillus nidulans reproduces asexually via uninucleate, haploid spores, which are produced on morphol. differentiated aerial structures, called conidiophores. These consist of four distinct cell types, a foot with a terminally swollen stalk, metulae, phialides and conidiospores. The mol. mechanisms underlying the morphol. changes that occur during conidiophore development have been studied by mutant anal. We have isolated the hymA mutant, in which conidiophore development is affected at the metula stage. In the mutant metulae do not differentiate properly but come to resemble hyphae (hym = hypha-like metulae). In this paper we have analyzed the corresponding gene. It encodes a highly expressed 44 kDa protein which resides in the cytoplasm and has homologues in yeast, plants, fly, worm, fish, mice and man. We constructed hym deletion strains of Saccharomyces cerevisiae and of A. nidulans and found that the gene is essential in S. cerevisiae but is dispensable in the filamentous fungus. A cellular function for the Hym protein has not yet been defined in any organism. To demonstrate functional conservation we constructed a chimeric protein comprised of the N-terminal half of the A. nidulans and the C-terminal half of the mouse homolog MO25. This hybrid protein could fully substitute for HymA function in A. nidulans. In addition, the mouse protein itself partially rescued the hymA mutation in the fungus. HymA is thus highly conserved in evolution and probably serves similar functions. The fact that hymA is required for conidiophore development in A. nidulans suggests that homologous genes in other organisms might also be involved in morphogenesis.
RE.CNT 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 16 OF 16 MEDLINE on STN DUPLICATE 7
AN 1996268479 MEDLINE
DN PubMed ID: 8672247
TI Molecular characterization of the Drosophila Mo25 gene, which is conserved among Drosophila, mouse, and yeast.
AU Nozaki M; Onishi Y; Togashi S; Miyamoto H
CS Research Institute for Microbial Diseases, Osaka University, Osaka, Japan.
SO DNA and cell biology, (1996 Jun) Vol. 15, No. 6, pp. 505-9.

Journal code: 9004522. ISSN: 1044-5498. L-ISSN: 1044-5498.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals
OS GENBANK-AB000402
EM 199608
ED Entered STN: 22 Aug 1996
Last Updated on STN: 3 Mar 2000
Entered Medline: 12 Aug 1996
AB To study the general physiological role of the Mo25 gene, which has been cloned from mouse cleavage-stage embryos, we isolated a Drosophila equivalent, dMo25, cDNA from an embryo cDNA library. The 2,222 nucleotides contained a single open reading frame encoding a polypeptide of 339 amino acid residues with a calculated molecular mass of 39,278 daltons. The deduced amino acid sequence of the dMo25 cDNA had 69.3% identity with mouse Mo25. A homology search revealed that these were similar to a protein encoded in an open reading frame near the calcineurin B subunit gene on chromosome XI in Saccharomyces cerevisiae. In particular, the carboxy-terminal region was highly conserved in Drosophila, mouse, and yeast. The dMo25 gene was mapped to the left arm of the third chromosome at 73AB, and 2.3- and 1.8-kb mRNA bands were detected during development and in adult Drosophila. Conservation of the gene structure and the wide expression profile indicated that the function of the gene is likely to be fundamental in many cell types as well as during development.

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